An Examination of Subjective Response to Alcohol in African Americans*

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ABSTRACT. Objective: Alcohol response is a widely studied risk factor for heavy drinking behavior and alcohol-use disorders. This study examined acute subjective response to alcohol as a predictor of drinking behavior, alcohol-related problems, and family history of alcoholism in African Americans. The convergent validity of self-reported response to alcohol (Self-Rating of the Effects of Alcohol scale [SRE]) in an African-American sample was also examined. **Method:** One hundred and three African-American young adults participated in an alcohol-challenge study, receiving a moderate dose of alcohol (0.72 g/kg alcohol for men, 0.65 g/kg for women). Breath alcohol concentration and subjective response to alcohol were assessed before beverage consumption, in 15-minute intervals for the first hour following consumption, and in 30-minute intervals thereafter. **Results:** Latent variable growth models indicated that experiencing increased acute stimulation from alcohol was related to past-month drinking behavior and alcohol-related problems. Regression analyses indicated that the SRE was related to drinking behavior, alcohol-related problems, having an alcohol-use disorder, and a family history of alcoholism. The SRE was not related to either sedation or stimulation following alcohol administration. **Conclusions:** Results support alcohol response as a marker of risk for increased drinking behavior and alcohol-related problems in African Americans. Further research is required to directly compare African-American and white response to alcohol within an alcohol-challenge paradigm. (*J. Stud. Alcohol Drugs* **70:** 288-295, 2009)

A LARGE BODY OF RESEARCH has shown that an individual's response to alcohol serves as a marker of risk for alcohol-use disorders and alcohol-related consequences. Individual differences in response to alcohol have been shown to influence risk for alcohol dependence (Schuckit and Smith, 2001) and to relate to increased heavy alcohol use (Conrod et al., 2001). Alcohol response has also been found to be heritable (Heath and Martin, 1991; Heath et al., 1999; Viken et al., 2003) and to differentiate between children of alcoholics and controls (Schuckit, 1985; Schuckit and Gold, 1988). These findings lend support to the construct of alcohol response as an endophenotype for alcohol-use disorders (Schuckit et al., 2007). The present study examined subjective response to alcohol as a marker of alcoholism risk in African Americans.

Considerable research evidence shows that the alcoholism risk process differs for African Americans and whites. Results from the National Comorbidity Survey Replication data found that the lifetime prevalence of alcohol-use disorders was lower for African Americans (9.5%) compared with whites (13.4%) and that African Americans have a lower lifetime risk of developing a substance-use disorder (Breslau et al., 2006). Additionally, in college populations, African Americans report lower rates of heavy drinking compared with whites (O'Malley and Johnston, 2002). However, despite lower rates of alcohol-use disorders, there is some evidence for increased persistence of alcohol dependence and more frequent engagement in heavy drinking occasions in African Americans in mid-adulthood (Dawson, 1998).

Because of the ethnic differences in polymorphisms of the alcohol metabolizing enzymes (alcohol dehydrogenase [ADH] and aldehyde dehydrogenase [ALDH]), response to alcohol has been studied across a variety of racial groups (e.g., Asian: Luczak et al., 2002; Native American: Garcia-Andrade et al., 1997; Latino: Schuckit et al., 2004). However, despite recent findings showing that *ADH* polymorphisms (e.g., *ADH1B*3*) may be linked to decreased risk for alcoholism in African Americans (Ehlers et al., 2001, 2003), studies have not examined how response to alcohol relates to drinking behavior in this group.

Individual differences in acute response to alcohol are frequently assessed through alcohol-challenge studies, where participants report on their subjective feelings of intoxication and/or physiological measures of response are obtained (e.g., heart rate) following a specified dose of alcohol. Such studies provide a fairly direct assessment of response and also allow for the examination of alcohol response on both the ascending and descending limbs of the blood alcohol curve. In their review and synthesis of the literature, Newlin and Thompson (1990) argued for the importance of examining response separately for the ascending and descending limbs of the blood alcohol curve. Alcoholism risk would be as-

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sociated with dampened response to the sedating effects of alcohol (King et al., 2002; Schuckit, 1980, 1984; Schuckit and Smith, 2001) on the descending limb and with increased response to the stimulating effects (Conrod et al., 1995, 2001; Erblich and Earleywine, 2003; Finn et al., 1990; King et al., 2002) on the ascending limb.

Despite the advantages of the alcohol-challenge paradigm, such studies are time consuming, expensive, and—in the United States—restricted to individuals age 21 or older. Schuckit and colleagues (1997a) developed a self-report measure designed to assess response to alcohol (Self-Rating of the Effects of Alcohol scale [SRE]) that can be used when alcohol-challenge studies are not feasible. Recently, Schuckit and colleagues (2007) showed that the SRE prospectively predicts alcohol-related problems and heavy use. One goal of the present study is to test the convergent validity of the SRE in an African-American sample, demonstrating that it is associated with acute response to alcohol in an alcohol challenge, as well as self-reported drinking behavior and a family history of alcoholism.

The present study had two primary aims. Our first aim was to test the association between acute subjective response to alcohol and drinking behavior in African Americans. We hypothesized that increased stimulation from alcohol on the ascending limb and diminished sedation on the descending limb would be related to having higher levels of drinking, experiencing alcohol-related problems, meeting lifetime criteria for an alcohol-use disorder, and having a family history of alcoholism. Our second aim was to examine the convergent validity of the SRE in an African-American sample. We hypothesized that higher scores on the SRE (i.e., reporting drinking more drinks to experience the effect) would be associated with having increased drinking behavior, experiencing decreased acute sedation on the descending limb of the breath alcohol curve, experiencing alcohol-related problems, having an increased likelihood of an alcohol-use disorder, and having a family history of alcoholism. Based on the content of the SRE items (e.g., passing out, slurring speech), we hypothesized that the SRE would not be related to acute stimulation on the ascending limb of the breath alcohol curve.

Method

Participants

Participants were 103 African-American young adults. The sample was 45% male and had a mean (SD) age of 21.83 (1.13) (range: 21-26). The majority of participants described themselves as being primarily of African-American descent; 3% of the sample identified themselves as Hispanic, and 7.8% of the sample described themselves as mixed race. To meet inclusion criteria for the study, those who identified as Hispanic or mixed race were required to have at least one biological parent who was African American. The majority of the sample (79%) had some college education, and 19% reported being college graduates. Participants were required to be between the ages of 21 and 26 and to be current drinkers. Additionally, participants were excluded if they were currently an abstaining alcoholic, had significant medical or psychiatric illness (e.g., psychotic disorders, past head injury with loss of consciousness > 5 minutes), or were currently taking medication for which use of alcohol is contraindicated.

Procedures

Study procedures were approved by the University of Missouri's Institutional Review Board. Participants were recruited from the University of Missouri and the city of Columbia, MO, and the surrounding area (Boone County, MO). Fliers were placed at various locations at the University of Missouri and at local businesses. Potential participants received a basic phone screening to determine eligibility.

Participants who met eligibility criteria were scheduled for an interview. On the scheduled interview day, participants were provided with an informed consent form to sign. Interviews, study tasks, and questionnaire completion were conducted in a private office. Participants received $40 for their participation in the interview.

Participants were then scheduled for a second appointment. Appointments were scheduled approximately 1 week after the initial interview. Participants were given an information packet before their laboratory appointment. They were instructed to refrain from alcohol for 24 hours before the session and to refrain from other drug use for 48 hours. They were also instructed not to eat or drink any fluids other than juice or water (e.g., no caffeinated beverages or dairy products) for 8 hours before their session (starting at midnight the evening prior). Participants who were taking medication that is safe to use while consuming alcohol but could affect subjective or physiological assessments (e.g., pseudoephedrine) were asked not to take the medication before the alcohol-challenge session.

The alcohol-challenge session took approximately 5 hours. Participants arrived at the laboratory at 8:00 AM. A questionnaire was administered to verify compliance with pre-session instructions. A breath alcohol concentration (BrAC) analysis was used to verify abstinence from alcohol. Women were given a urine pregnancy test and excluded from the study if they tested positive. During the baseline period, a low-fat breakfast was provided.

Between 8:30 AM and 9:00 AM, baseline measures were taken. At 9:00 AM, participants received an alcoholic beverage (vodka and tonic; range of total volume consumed: 352 ml-1,241 ml). Participants received a dose of alcohol equivalent to 0.72 g/kg alcohol for men and 0.65 g/kg alcohol for women, designed to reach a peak blood alcohol

concentration of approximately .075 mg%-.080 mg% (Sher and Walitzer, 1986). Dosing was based on weight and gender (range of total vodka consumed: 70 ml-248 ml). The alcohol drinks were made using 50% alcohol (vodka) in 20% solution with noncaffeinated soda (tonic). Beverages were consumed over a 15-minute period.

Participants were assessed before beverage consumption, in 15-minute intervals for the first hour following consumption, and in 30-minute intervals thereafter. The alcohol administration and assessment were conducted in a private office, using a semirecumbent chair, separate from that used for interviews. The office was equipped with a vital signs monitor and a computer. At approximately noon, each participant was provided lunch. To minimize risk, the following procedures outlined in the National Institute on Alcohol Abuse and Alcoholism (2005) Recommended Council Guidelines on Ethyl Alcohol Administration in Human Experimentation were used. Participants were not allowed to leave the laboratory until their observable behavior had returned to normal and until their BrAC fell below .02%. Each participant was also required to travel home by taxi (provided by the study) or with a friend. Participants were required to state in writing that they would not drive a car or operate other machinery for at least 3 hours after leaving the laboratory. They were reimbursed $100 for participation in the session.

Measures (interview session)

Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA-II). This clinical interview was used to collect demographic information, medical history, substance abuse and dependence symptoms, and alcohol and substance-use behaviors. The SSAGA has been found to be a reliable and valid instrument for the assessment of numerous psychiatric disorders, including alcohol dependence (Bucholz et al., 1994; Hesselbrock et al., 1999). The Family History Assessment Module assessed first-degree family history of alcohol dependence (Rice et al., 1995).

Alcohol-use behavior. The Drinking Styles Questionnaire (DSQ; Smith et al., 1995) was used to provide additional information on alcohol involvement. The DSQ collects information about drinker/nondrinker status, age at first drink, past-month and typical frequency and quantity of drinking, frequency of drunkenness, problems perceived to be caused by one's drinking, and typical drinking contexts. The DSQ has demonstrated good reliability and validity in adolescent samples (Smith et al., 1995) and has been used in white and African-American college samples (McCarthy et al., 2001).

Two separate dependent variables were created using items from the DSQ. A past-month drinking variable included quantity, frequency, number of times having five or more drinks, and largest number of drinks consumed on one occasion. For growth curve analyses, a latent variable model with these variables as indicators was used as a dependent measure, whereas factor scores were used for regression analyses. A second dependent variable, alcohol-related problems, was created by summing eight true/false items that assessed negative consequences of alcohol use (e.g., experienced legal problems, blackouts). The DSQ contains 10 true/false items that are designed to look at alcohol-related problems (e.g., blackout, trouble with friends, legal problems). We excluded two items (felt nauseous, had a hangover) because of lack of variability in our sample (>80% of participants endorsed these items).

Retrospective response to alcohol. The SRE (Schuckit et al., 1997a) was used to assess retrospective sensitivity to alcohol. This scale asks participants to report the number of drinks required to feel the effects of alcohol (i.e., feel different, feel a bit dizzy or slur speech, begin stumbling, pass out) the first five times the participant drank, after 3 months of drinking at least once a month, and during the period of heaviest drinking. Schuckit and colleagues (1997a) found the SRE to be a valid indicator of subjective response to alcohol in adults.

Measures (alcohol-challenge session)

Breath alcohol concentration. BrAC readings were taken using a breath alcohol analysis device (Alco-Sensor, FST; Intoximeters, Inc., St. Louis, MO) at baseline and at all measurement points (i.e., 15, 30, 45, 60, 90, 120, and 150 minutes) after consumption of the beverage.

Subjective feelings of intoxication. Subjective feelings of intoxication were evaluated at baseline and at all measurement points following beverage consumption using the Biphasic Alcohol Effects Scale (BAES; Martin et al., 1993). This measure assesses separate sedating and stimulating effects of alcohol on both the ascending and descending limbs of the breath alcohol curve. This measure has been used to discriminate sedating and stimulating effects of alcohol in alcohol-challenge studies and has a four-factor structure (Type of Response × Blood Alcohol Curve Limb; Earleywine and Erblich, 1996).

Data analytic plan

SPSS 14.2 (SPSS, Inc., Chicago, IL) and Mplus 5.1 (Muthén and Muthén, 2008) were used to conduct statistical analyses for the current study. All study analyses controlled for gender. As subjective response during alcohol challenge is a repeated measurement, latent variable growth models were used to account for the nesting of multiple assessments within person. Latent growth models were used to test hypotheses that experiencing increased stimulation on the ascending limb and decreased sedation on the descending limb of the breath alcohol curve is associated with increased drinking behavior, experiencing alcohol-related problems,

meeting lifetime criteria for an alcohol-use disorder, and having a family history of alcoholism. For the ascending limb, BAES stimulation at baseline and at 15, 30, and 45 minutes served as indicators of stimulation, whereas for the descending limb, BAES sedation at 60, 90, 120, and 150 minutes served as indicators of sedation. The slope parameter was used to predict drinking behavior, alcohol-related problems, and having an alcohol-use disorder. Time was coded so that the intercept reflected the model-implied baseline assessment (baseline for stimulation, 60 minutes for sedation). For stimulation and sedation, the intercept latent variable was set to predict the slope latent variable to control for baseline differences in stimulation/sedation. Past-month alcohol use was modeled as an endogenous latent variable, with the slope parameter of alcohol response specified to predict the alcohol-use latent variable.

To examine our second study aim, testing the convergent validity of the SRE in an African-American sample, regression analyses examined the association between the SRE, drinking behavior, alcohol-related problems, meeting lifetime criteria for an alcohol-use disorder, and having a family history of alcoholism after controlling for gender. Three different composite SRE variables were computed (i.e., the first five times the participant drank, after 3 months of drinking at least once a month, and during the period of heaviest drinking). These SRE variables were used as predictors of drinking behavior, alcohol-related problems, meeting criteria for alcohol-use disorder (according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition [DSM-IV-TR]; American Psychiatric Association, 2000), and having a family history of alcoholism. Additionally, regression analyses, controlling for baseline sedation levels and gender, were used to test the hypothesized association between the SRE variables and sedation at four separate time points on the descending limb (i.e., 60, 90, 120, and 150 minutes after drinking). Similar analyses tested the association between SRE variables and stimulation at three separate time points on the ascending limb (i.e., 15, 30, and 45 minutes after drinking).

Results

Descriptive information about alcohol use

Table 1 presents mean levels of age at first drink and past-month drinking behavior separately by gender. *T* tests were used to test mean level gender differences in drinking behavior. Analyses showed that men reported drinking alcohol more frequently, consuming at least 5 drinks on more occasions, and consuming a larger number of drinks on a single occasion than did women. Men and women did not differ in age at first drink or past-month average quantity of drinks consumed. Four participants met DSM-IV-TR diagnostic criteria for lifetime alcohol dependence (3.9%) and 25 participants met criteria for lifetime alcohol abuse (24.3%).

Table 1. Descriptive statistics for alcohol-use variables

Variable	Men Mean (SD)	Women Mean (SD)
Age at first drink	15.13 (3.52)	16.75 (2.81)
Frequency	8.96 (9.12)†	5.02 (4.62)
Quantity	3.65 (1.58)	2.55 (1.29)
≥5 drinks	3.72 (4.25)‡	1.05 (1.77)
Largest no. of drinks	7.37 (4.38)†	4.11 (2.21)

Notes: Values are means and standard deviations of raw scores. *T* tests were used to compare mean level differences between men and women.
†$p < .01$; ‡$p < .001$.

Acute response to alcohol

We first estimated latent variable growth models for stimulation on the ascending limb and sedation on the descending limb. Using the cutoffs outlined in Hu and Bentler (1999), model fit indices indicated acceptable fit for the stimulation model ($\chi^2 = 26.37$, 5 df, $p < .001$; $n = 103$; comparative fit index [CFI] = .92) and good fit for the sedation model ($\chi^2 = 14.26$, 5 df, $p < .05$; $n = 103$; CFI = .97). We then estimated models with gender predicting growth in stimulation and sedation (i.e., slope parameters). Results from these models indicated that men experienced increased stimulation from alcohol on the ascending limb (β [SE] = .32 [.12], $p < .01$), but men and women did not differ in sedation on the descending limb (β = .03 [.09], NS).

Next, we used the slope parameters of stimulation and sedation to predict past-month self-reported drinking behavior. Results indicated that changes in stimulation on the ascending limb were significantly associated with past-month drinking behavior (β = .39 [.14], $p < .01$; see Figure 1). Changes in self-reported sedation on the descending limb were marginally associated with past-month drinking (β = .25 [.13], $p < .05$, one tailed).

We also tested whether changes in stimulation and sedation predicted alcohol-related problems and meeting lifetime criteria for an alcohol-use disorder. Changes in stimulation on the ascending limb were related to reporting higher levels of alcohol-related problems (β = .23 [.11], $p < .05$), whereas experiencing decreased sedation on the descending limb was not (β = .08 [.12], NS). Change in stimulation on neither the ascending limb (β = .18 [.16], NS) nor descending limb (β = −.08 [.14], NS) was related to having an alcohol-use disorder.

Finally, we tested family history of alcoholism as a predictor of acute response to alcohol. Family history in either first-degree (mother/father) or second-degree (e.g., aunt/uncle, grandparent) relatives was not related to experiencing increased stimulation (first degree: β = −.05 [.12], NS; second

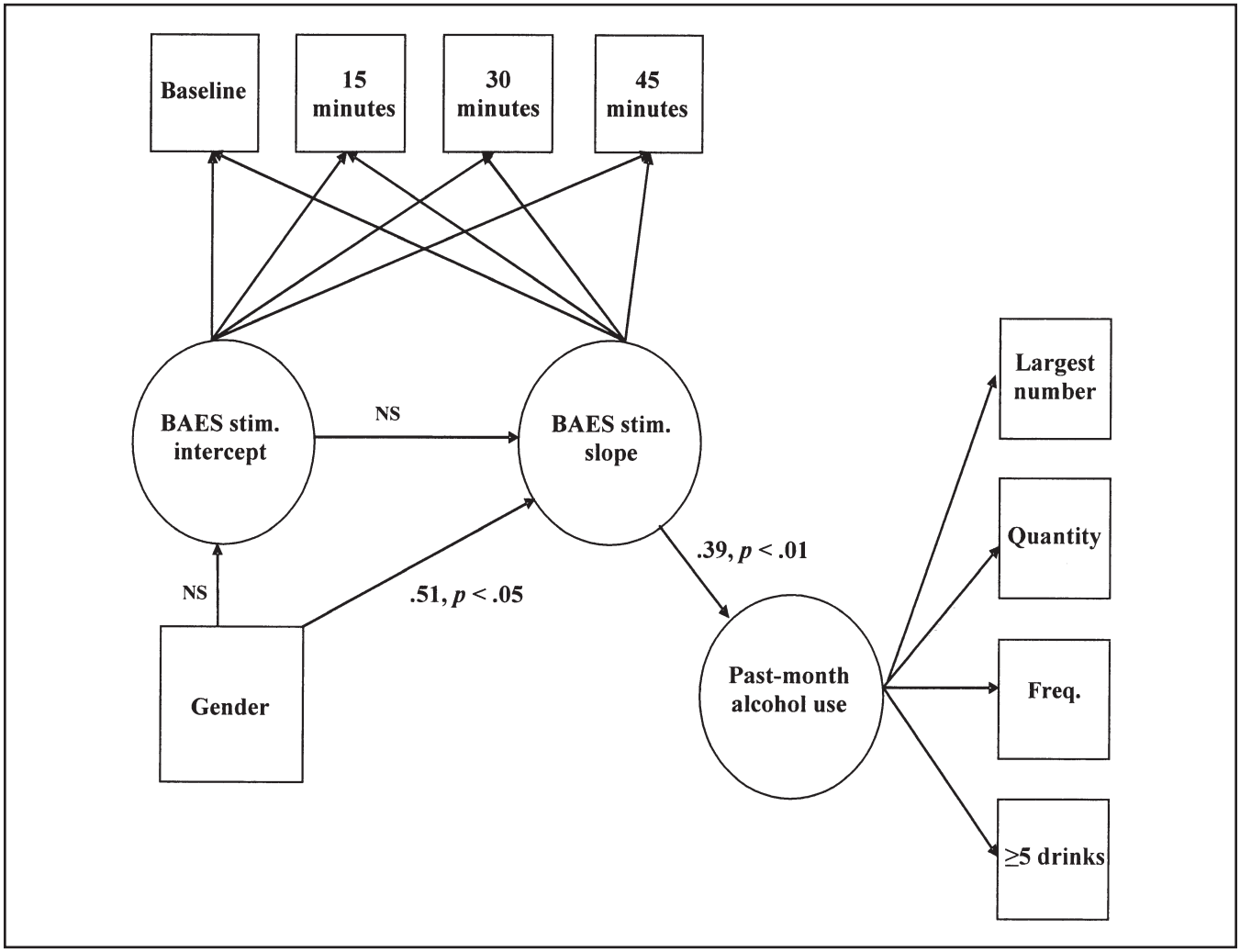

FIGURE 1. Biphasic Alcohol Effects Scale (BAES) stimulation (stim.) predicting past-month drinking behavior. The figure represents the model testing the association between BAES stimulation and past-month alcohol use. Experiencing increased stimulation on the ascending limb was associated with past-month drinking behavior ($p < .01$). For ease of presentation, factor loadings and error terms are not depicted. Freq. = frequency; NS = not significant.

degree: β = .07 [.12], NS) on the ascending limb or decreased sedation (first degree: β = .01 [.04], NS; second degree: β = .10 [.01], NS) on the descending limb.

Convergent validity of the Self-Rating of the Effects of Alcohol scale

We examined the association between the SRE and self-reported past-month drinking behavior and alcohol-related problems, controlling for gender. Regression analyses indicated that past-month drinking behavior was associated with initial low sensitivity (β = .28 [.09], $p < .01$), low sensitivity after 3 months of drinking at least once a month (β = .39 [.09], $p < .001$), and low sensitivity during the period of heaviest drinking (β = .53 [.09], $p < .001$). Additionally, results for alcohol-related problems were associated with low sensitivity after drinking for 3 months (β = .23 [.11], $p < .05$) and during the period of heaviest drinking (β = .33 [.11], $p < .01$) but not with initial low sensitivity (β = .11 [.11], NS). Logistic regression analyses with alcohol-use disorder as the dependent variable closely paralleled these results. Low sensitivity after drinking for 3 months (odds ratio = 1.40 [0.15], $p < .05$) and during the period of heaviest drinking (odds ratio = 1.34 [0.11], $p < .01$) were related to meeting lifetime criteria for an alcohol-use disorder, whereas initial low sensitivity was not (odds ratio = 0.99 [0.16], NS).

We then tested the association between family history and the SRE, again controlling for gender. Results indicated that low sensitivity to alcohol after 3 months of drinking at least once a month was related to having more second-degree relatives with alcohol-related problems (β = .24 [.11], $p < .05$) but not first-degree relatives (β = .10 [.12], NS). Initial low sensitivity (first degree: β = .13 [.11], NS; second degree: β = .15 [.11], NS) and low sensitivity during the period of heavi-

est drinking (first degree: β = .07 [.12], NS; second degree: β = .11 [.12], NS) were not related to family history.

Finally, we tested the association of the SRE with acute stimulation and sedation on the ascending and descending limbs of the breath alcohol curve while controlling for baseline stimulation/sedation levels and gender. The SRE variables were not related to acute stimulation (β's: −.04-.10, NS) on the ascending limb of the BrAC curve or acute sedation (β's: −.07-−.18, NS) on the descending limb of the BrAC curve.

Discussion

Results of the current study provide support for subjective response to alcohol, both retrospective and acute, as a marker of risk for increased drinking behavior in African Americans. For acute response, experiencing increased stimulation on the ascending limb was related to increased drinking behavior and alcohol-related problems. There was also a modest association between sedation on the descending limb and drinking. However, acute response to alcohol on the ascending and descending limbs was not related to having an alcohol-use disorder. The SRE, a retrospective self-report measure of alcohol response, was related to increased drinking, alcohol-related problems, and meeting criteria for a lifetime alcohol-use disorder. These findings are largely consistent with previous studies conducted on whites that have shown that increased stimulation (physiological and subjective) on the ascending limb is related to heavy drinking (Conrod et al., 2001; Erblich and Earleywine, 2003; Finn et al., 1990; King et al., 2002) and low sensitivity as assessed by the SRE is related to increased risk for alcohol problems (Schuckit et al., 2007).

However, some findings differed from previous research on alcohol response. First, in this sample, the SRE and acute response were unrelated. This is inconsistent with previous research on the SRE (Schuckit et al., 1997b) that has been validated as a measure of response to alcohol through correlations with acute response. However, prior studies validating the SRE have examined its association with the Subjective High Assessment Scale (SHAS: Schuckit and Gold, 1988) as a measure of acute response to alcohol. Although the SHAS items (e.g., discomfort, high, clumsiness, confusion) seem to parallel the items of the sedation subscale of the BAES (e.g., difficulty concentrating, heavy head, sluggish, inactive), the two measures may assess different aspects of acute response to alcohol. Future studies could use the SHAS in an African-American sample to examine this possibility. Given that both the SRE and changes in stimulation in the alcohol challenge were related to drinking behavior, it may be that in this sample these two measures assess discrete constructs related to risk for heavy alcohol use. However, in the current study, the SRE was related to drinking behavior, alcohol-related problems, and alcohol-use disorders, whereas acute response to alcohol (BAES) was not related to alcohol-use disorders. This may indicate that, for African Americans, acute response to alcohol is associated with increased alcohol use but not alcohol-use disorders.

Another difference in the current results is that differences in response to alcohol, assessed through either the SRE or alcohol challenge, were largely unrelated to family history of alcoholism. The one exception was the SRE assessment of low sensitivity to alcohol after drinking at least once a month for 3 months, which was related to the number of second-degree relatives with alcohol problems. Previous research on response to alcohol and family history has primarily been conducted with white samples and has been oversampled to contain a larger number of family history–positive participants (Newlin and Thompson, 1990). Although we assessed family history in the present study, our sample was not recruited to have equal numbers of participants with and without a family history of alcoholism. Low base rates of individuals with a first-degree relative (n = 14) may have reduced our ability to detect the association between alcohol response and family history. Additionally, Garcia-Andrade and colleagues (1997) did not find differences in subjective response to alcohol depending on family history status in a Native American population. It may be that response does not differ as a function of family history for African Americans. Future studies could address this possibility by increasing recruitment of family history–positive African Americans within an alcohol-challenge paradigm.

Although results showed that subjective response to alcohol functioned similarly for African Americans as has been found in white samples (Erblich and Earleywine, 2003; Schuckit et al., 2007), a limitation of the current study is not directly comparing African-American and white response to alcohol. The clearest test of potential differences in alcoholism risk factors between African Americans and whites would be by comparing both groups within the same alcohol-challenge paradigm.

Another limitation of the present study is the restriction of the sample to current drinkers (a requirement of this type of alcohol-challenge study). Limiting the sample to current drinkers restricts the range of drinking variables, which can reduce observed associations with drinking. Furthermore, because this is a cross-sectional study of current drinkers, there is no empirical basis for the temporal sequencing of study variables. Our sample also contained individuals who already reported having a lifetime diagnosis of an alcohol-use disorder, which limits our ability to determine if response to alcohol is a risk factor for alcoholism or a result. We have interpreted these findings within the theoretical framework that posits level of response to alcohol as a risk factor for alcohol-use disorders (Schuckit and Smith, 2001), and this is consistent with the format of the SRE, which asks participants to retrospect about their response to alcohol at an earlier period. Longitudinal studies, including individuals who

make the transition to drinking, are required to test whether level of response predicts later alcohol use and alcohol-use disorders. This may be particularly relevant given our findings that acute response to alcohol was not associated with alcohol-use disorders. A final limitation is that our study had a high percentage of student participants. Although attempts were made at recruiting a community sample, the majority of our participants were college students, which may limit the generalizability of our results.

African Americans have a different drinking topography than whites (Breslau et al., 2006; Dawson, 1998). The current study demonstrated that one widely studied risk factor for white drinking, response to alcohol, is related to increased drinking and alcohol-related problems in African Americans. Results of this study suggest that response to alcohol can serve as a marker of risk for African Americans, and future studies can incorporate this construct into models of risk and protective factors for African-American drinking.

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